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**Induction of SOS response by 9-aminoacridine does not much, if any at all, influence frameshift mutagenesis of *Escherichia coli***

The acridine dye 9-aminoacridine (9-AA) is thought to mainly cause frameshift mutations. It is, however, unclear whether the frameshift mutagenesis induced by 9-AA is dependent upon the SOS response regulated by *recA* and *lexA* genes. In order to understand the genetic effects of 9-AA, we have examined the induction of SOS regulon expression. W-reactivation of UV-irradiated  $\lambda$  phage and the frameshift mutagenesis by 9-AA using the *Escherichia coli* strains with different DNA repair capacities. 9-AA at concentrations ranging from 5 to 30  $\mu\text{g/ml}$  induced both *recA* and *umuC* gene expression in the wild-type, *recA* and *lexA* mutant strains. The plating efficiencies of UV-inactivated  $\lambda$  phage were increased with increasing concentrations of 9-AA not only in the wild-type but also in the *recA*, *lexA* and *umuC* mutants. *TrpE* and *nad* frameshift alleles in the wild-type, *recA*, *lexA* and *umuC* genetic backgrounds examined were effectively reverted by 9-AA in a dose-dependent manner. Sensitivity to killing by 9-AA was identical in the presence and absence of the *recA*<sup>+</sup> and *lexA*<sup>+</sup> genes. The fact that frameshift mutagenesis is induced in the *recA* and *umuC* mutants by 9-AA suggests that induction of the SOS response by 9-AA does not much, if any at all, influence frameshift mutagenesis.

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**Detection of the cytogenetic effect of inhaled cigarette smoke by the micronucleus test**

The induction of micronuclei in mice exposed to cigarette smoke by inhalation was examined. The smoke was generated from the Hamburg II smoking machine with 30 cigarettes. The mainstream and sidestream of smoke were sep-

arately given to mice once a day, for 28 days. Control mice received exposures to fresh air as a sham-smoke. After 1 or 2 days of smoke exposure, there was no significant difference in the frequency of micronucleated polychromatic erythrocytes (MNPCEs) in bone marrow between controls and the smoke-exposed animals. On the contrary, slight increases in the frequency of MNPCEs were observed in mice exposed to cigarette smoke for 3 days; the control incidence of MNPCEs per 1000 polychromatic erythrocytes was 1.2, while in mice exposed to the sidestream or mainstream of smoke the incidences were 2.8 or 1.8 respectively. Continued daily exposure for 4, 11, 18 and 25 additional days showed no further increase in the frequency of MNPCEs. The results of this study suggest that there is a difference in the cytogenetic activity between the sidestream and mainstream of cigarette smoke.

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**Screening of antimutagenic activities of herbal extracts**

Antimutagenic activity of approximately 50 herbal extracts which have been used as 'old wives' remedies' was examined. Inactivation of 4NQO mutagenicity (series i) and suppression of UV mutagenesis (ii), both in bacteria, were used as the assay system. For the mutagenesis, the reverse mutation in *S. typhimurium* TA98 and TA100, and the mutation of *E. coli* WP2 uvrA/pKM101 to tryptophan prototroph (a reversion) and to low-concentration streptomycin resistance (a forward mutation) were used. The extract samples were obtained by extraction with distilled water or ethanol. For experimental series i, a mixture of the extract and 4NQO was incubated and then plated with the bacteria. For series ii, the UV-irradiated bacteria were plated with the extract sample. When the number of mutant colonies decreased more than 50% by adding the extracts, the antimutagenic activity was judged as positive.

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